PREFACE

To all our friends,,

To all these persistent, determined, hard worker and cheerful persons ...

To all these souls who set up their goals to relieve pain of people ...

To all spectacular , dedicative , upcoming doctors Insha'Allah ...

We prepared this book, hoping to be a step in your way to success ...

In your way to reach the summit together.

Looking forward to help you achieving the full-mark in Pathology exams.

Welcoming any suggestions, feedback or complain.

Notice also that this edition contains answers of MCQs exams.

Special Thanks to dr MARWA NOOR for her great efforts in this book.

May Allah bless her all the time.

BEST WISHES

IBNKHALDON MED TEAM

Written Exams & Answers of Pathology | Ibnkhaldon Med

INDEX

Idea of the book!

This book contains full answers of written exams in pathology from 2008 to 2014, and some questions before that date even!

Answers were arranged in a way that makes the student comfortable with, and we tried to make the book well categorized and designed.

This book is of a great value to be studied and feel free to dig deeply in.

Cardiovascular System

MAY 2008, 2009

[1] Discuss cardiac pathological lesions in rheumatic fever and mention the possible complications.

➢ Cardiac pathological lesions: "pancarditis"

a- **Pericardium :**

- Serous or serofibrinous pericarditis in the form of fibrin threads on both pericardial layers giving a " bread and butter " appearance on separation.
- Fate: fibrinolysis or fibrosis with diffuse adhesion or localized (milk spots).

b- Myocardium:

- Myocarditis
  - **Gross:** enlarged heart , flabby with scattered pin head size pale grayish foci which appear after four weeks.
  - **E/M:**

    Aschoff nodules "pathognomonic lesion of this disease".
    - ✓ Site: within the interstitial connective tissue between the muscle fibers and in relation to blood vessels
    - ✓ Composition: necrosed collagen with fibrin deposition , surrounded by lymphocytes , macrophages , modified histiocytes (Aschoff giant cells) and modified mesenchymal cells (antischow ceels or caterpillar cells)

c- **Endocardium :**

- Endocarditis :
  - ✓ Mural endocardium: inflammation of the endocardium lining the cardiac chambers especially that of left atrium, which is :
    - Swollen , oedematous and red.
    - Thrombosis due to erosion by the flowing blood.
    - (MacCallum's patch): focus of rough wrinkled endocardium of the posterior aspect.
  - ✓ Inflammation of Valvular endocardium (Rheumatic Valvulitis)

1- **Acute :**

- Gross :
  - Valves are swollen , congested and oedamatous
  - Specially : mitral and aortic valves

***Rheumatic vegetations :**

- Gross :
  - ✓ Site :
  - along the line of closure 2-3 mm from the free margin
  - Firmly adherent to the atrial surface of the mitral valve or ventricular surface of aortic valve
  - ✓ Shape : multiple beaded thrombi
  - ✓ Size : pin head size
  - ✓ Never emboli
- Microscopic :
  - ✓ Aseptic and formed of platelets and fibrin with lymphocytes , plasma cells and aschoff giant cells in the subendocardium

2- **Chronic rheumatic valvulitis :**

- Fibro calcific thickening with intercommissural adhesions of the involved valve leading to marked stenosis (fish mouth or button hole) or regurgitation
- Cordae tendenae :
  - Thickened
  - Fused
  - Shortened

  :Leading to funnel shape appearance of the valve

➢ **Possible complications :**

- Acute heart failure
- Congested lung and pulmonary hypertension
- Embolism from mural thrombus
- Infective endocarditis

JUNE 2013

[2] Aschoff,s nodule is formed of ……,……,……..

1- Necrosed collagen with fibrin deposition, surrounded by lymphocytes, macrophages.
2- Modified histiocytes (Aschoff giant cells).
3- Modified mesenchymal cells (antischow cells or caterpillar cells).

MIDYEAR 2006

[3] Define giving one example: endocarditis.

➢ Def.: Inflammation Of mural or valvular endocardium.

➢ Example : acute bacterial endocarditis.

	ABE = Acute Bacterial Endocarditis
Organism	Highly virulent (can attack normal heart)
Age	50 – 60 y
Causes	- Pyogenic M/C is staph. aureus - Fungal if ↓ immunity
Gross	• Usually mitral & aortic valve → ulcer or perforation • Large bulky, globular vegetation • Friable, easily detachable → emboli → Pyaemic abscess
MP	- Acute suppurative inflam. + ulcer. - Vegetation = platelets + fibrin + bacteria
Fate & Comp.	• Local: cusp ulcer, perforation or rupture. • General: septic emboli → Pyaemic abscess Toxaemia, septicaemia, pyaemia

SEP 2013

[4] Compare: acute and sub-acute bacterial endocarditis.

	ABE = Acute Bacterial Endocarditis	SABE = Sub acute Bacterial Endocarditis
Organism	Highly virulent (can attack normal heart)	Low virulent
Age	50 – 60 y	Young & children
Causes	-Pyogenic M/C is staph. aureus -fungal if ↓ immunity	Non Pyogenic as strept. Viridans follow tooth extraction
Gross	• Usually mitral & aortic valve → ulcer or perforation • Large bulky, globular vegatation • Friable, easily detachable → emboli → Pyaemic abscess	• Usually mitral & aortic • Less bulky • Friable, easily detachable → sterile emboli → infarction
MP	- Acute suppurative inflam. + ulcer - Vegetation = platelets + fibrin + bacteria	- Fibroblastic proliferation - Vegetation = platelets + fibrin + polymorphs + bacteria
Fate & Comp.	• Local: cusp ulcer, perforation or rupture • General: septic emboli → Pyaemic abscess • Toxemia, septicaemia, pyaemia	• Worsening of pre-existing heart disease due to healing of the valvular lesions with scaring. • Sterile emboli → infarction or mycotic aneurysm in cerebral , superior mesenteric , renal and limb arteries. • Allergic vasculitis: →skin: splinter haemorrhage , Osler nodules → kidney: focal GN →Petechial haemorrhage in skin , retina ,

		serous sacs , conjuctiva and mucous membrane • General manifestation of toxaemia • Cause of death: CHF, RF, emboli, 2ry bacterial infection.

MAY 2010

[5] Compare between acute rheumatic valvulitis and sub acute bacterial endocarditis.

	Acute rheumatic valvulitis	SABE = Sub acute Bacterial Endocarditis
Organism	Group A β-hemolytic streptococci	Low virulent organism As strept. Viridans follow tooth extraction .
Age	5-15 years	Young & children
Gross	• Site: - Along the line of closure 2-3 mm from the free margin -Firmly adherent to the atrial surface of the mitral valve or ventricular surface of aortic valve - Valve is swollen , congest ed and oedamatous • Shape: multiple beaded thrombi • Size: pin head size • Never emboli	- Usually mitral & aortic valves: , • Less bulky than acute type • Friable, easily detachable → sterile emboli → infarction
MP	- Vegetation "aseptic" = platelets + fibrin + lymphocytes, plasma cells and aschoff giant cells in the sub endocardium.	- Fibroblastic proliferation of underlying valve. - Vegetation = platelets + fibrin + polymorphs + bacteria.

Fate & Comp.	- If persist → chronic rheumatic valvulitis: - Fibro calcific thickening with inter commissural adhesions of the involved valve leading to marked stenosis (fish mouth or button hole) or regurgitation - Cordae tendenae : • Thickened • Fused • Shortened Leading to funnel shape appearance of the valve - Acute heart failure - Congested lung and pulmonary hypertension - Embolism from mural thrombus - Infective endocarditis	• Worsening of pre-existing heart disease due to healing of the valvular lesions with scaring • Sterile emboli → infarction or mycotic aneurysm in cerebral , superior mesenteric , renal and limb arteries • Allergic vasculitis →Skin: splinter haemorrhage , Osler nodules → Kidney: focal GN →Petechial haemorrhage in skin , retina , serous sacs , conjuctiva and mucous membrane • General manifestation of toxaemia • Cause of death : CHF, RF, emboli, 2ry bacterial infection

SEP 2012, 2014

[6] Compare between vegetations of acute rheumatic valvulitis and acute bacterial endocarditis.

Discussed before

SEP 2008

[7] Enumerate types of endocarditis, discuss in details the pathology of subacute bacterial endocarditis.

- Types :
  1- Rheumatic endocarditis
  2- Infective endocarditis , which may be acute or sub-acute bacterial endocarditis
  3- Non-bacterial thrombotic endocarditis
  4- Atypical verrucous endocarditis
  5- Valvular lesions in carcinoid syndrome
- Pathology of sub-acute bacterial endocarditis :

  Discussed before

JUNE 2012

[8] Give reasons: rupture of infracted area of left ventricle commonly occurs 4-7 days after infarction.

- As the infracted area at this time is very weak due to:
  - Appearance of macrophages with early disintegration and phagocytosis of nuclear debris
  - Appearance of granulation tissue at the edge of infarction

SEP 2012

[9] Explain: the occurrence of myocardial infarction without occlusion of the stenosed arteries.

- May be explained as follows :
  1- Stenosed artery + excessive exertion lead to increased heart rate and decreased O2 flow with consequent myocardial infarction without thrombosis
  2- Reversible transient ischeamia + stenosed artery leads to impairment of myocardial contractility and myocardial infarction
  3- During night : slow heart rate + stenosed artery leads to diminished coronary perfusion followed by myocardial infarction

[10] Compare between: benign and malignant hypertension.

	Benign hypertension	Malignant hypertension
% Age	- Common - Above age of 50	- Rare - Mainly young adult
Course	-Slowly progress (years) - <200/100	-Rapidly progressive (fatal) -serious ↑e.g.280/180
Organs Affected	- **Heart:** lt. atrium &lt. vent. Hypertrophy - **Kidney:** benign nephrosclerosis = arteriolosclerotic = 1ry contracted kidney	↑ Headache, blurred vision, retinal he ± papilledema - **Heart:** usually normal size - **Retinal** dysfunction - **Kidney:** changes in distal interlobular & afferent arterioles → glomerulus → rupture →hematuria & Flea bite hges (malignant nephrosclerosis)
MP	Arterioles esp. kidney showing: -**Intimal thickening:** dt↑CT. -**Hyalinosis:** • Patchy then all circumference • Sub endothelial then all layers except endoth. - **Elastosis:** ↑elastic tissue	Arterioles esp. kidney showing: - **Fibrinoid necrosis**=necrosis+plasma constituent - **Endotheliosis:** • Concentric thick intima → endarteritis → narrowing • Best seen in interlobular aa of kidney

COD	-CHF45% -Coronary insuff. 45 % Cerebral (mainly hge) 5% -RF 5%	Poor prognosis -RF 95% -Cerebral (hge) -Coronary -CHF

MIDYEAR 2006

[11] Define aneurysm. Mention the different etiological types.

- Def: abnormal localized dilatation of an artery , vein or heart.
- Types:
  - a- True aneurysm : wall is formed by one or more layers of the affected vessels e.g. congenital aneurysm, syphilitic aneurysm, mycotic aneurysm.
  - b- False aneurysm: the wall is formed of connective tissue which is not a part of a vessel E.g. Pulsating hematoma, arteriovenous fistula.

MIDYEAR 2007

[12] Enumerate causes of right ventricular failure.

1- Chronic lung disease: -
   - Emphysema.
   - Ayerza's disease "bilharzial or syphilitic".
   - Fibrosis (TB, silicosis, bronchiectasis).
2- It. sided Heart Failure.
3- MS "mitral stenosis".
4- Pulmonary Stenosis or Pulmonary incompetence.
5- Congenital Heart Disease as Fallot's tetralogy &Ventricular Septal Defect.

JULY 2011

[13] List the complications in atherosclerosis.

1- The aorta:
   - a- Weaking of the wall which becomes dilated i.e. atheromatous aneurysm
   - b- Mural thrombosis may occur with detachment and embolic manifestations
2- In the medium sized :
   - a- Narrowing of the lumen leading to ischemia
   - b- Complete occlusion by thrombosis or hemorrhage or ulceration of the plaques leading to cut of blood supply (infarction)

JUNE 2013

[14] Compare between: true aneurysm and false aneurysm.

	True aneurysm	False aneurysm
Wall	Formed by one or more layers of the affected vessels	The wall is formed of connective tissue which is not a part of a vessel
Types .	1- Congenital aneurysm 2- Syphilitic aneurysm 3- Mycotic aneurysm 4- Atheromatous aneurysm 5- Dissceting aneurysm 6- Aneurysm of poly arteritis nodosa	1- Pulsating hematoma 2- Arteriovenous fistula

SEP 2013

[15] Complete: cause of death in aortic aneurysm is ……

- Haemopericardium with cardiac tamponade
- Mediastinal hemorrhage
- Haemothorax
- Abdominal hemorrhage depending on the site of rupture

SEP 2011

[16] A 10 year female complained of pharyngitis and after 2 weeks. She developed arthritis of the knee joint and the skin rash. Clinical examination showed tachycardia and pericardial rub.

a-What is the most likely diagnosis?

Rheumatic fever

b- What are the serological tests you recommend that help in the diagnosis of this case?

- Antistreptolysin O titre
- C reactive proteins in blood
- E.S.R

c-Explain the pathogenesis of the disease.

- Occur after a latent period of 2-3 weeks following a Respiratory infection by group A β-hemolytic streptococci
- M protein on surface of strept. Evoke immune reaction → production of antibodies that cross react with human cardiac muscle and valve components and joints of body.

d-What is the pathognomonic lesion of this disease?

Aschoff nodules.

- ✓ Site: within the interstitial connective tissue between the muscle fibers and in relation to blood vessels.
- ✓ Composition: necrosed collagen with fibrin deposition , surrounded by lymphocytes , macrophages , modified histiocytes (Aschoff giant cells) and modified mesenchymal cells (antischow ceels or caterpillar cells).

MIDYEAR 2012

[17] A male patient 50 years old complained of severe crushing chest pain radiated into the neck and shoulder. The pain lasted for several hours accompanied by dyspnea not relieved by nitroglycerin. The patient was hospitalized and investigation revealed elevated serum creatine kinase and serum tropinin one.

a- What is the most likely diagnosis?

Myocardial infarction

b- List three complications.

- Pericarditis.
- Arrhythmia.
- Heart Failure.
- Embolism.
- Rupture → tamponade.
- Fibrosis → aneurysm → embolism.

c- If this patient died after one week. Describe the histopathological changes that occur in the cardiac lesion at that time.

- Gross:- hyperemic edge
- Microscopic:
  - Appearance of macrophages with early disintegration and phagocytosis of nuclear debris
  - Appearance of granulation tissue at the edge of infarction

JUNE 2014

[18] Give reasons:

- Petechial hemorrhage in retina and conjunctiva in a patient with sub acute bacterial endocarditis, due- to allergic vasculitis- "- Type- III- hype- hypersensitivity"- which- considered- as- a- complication- of- SBE.

[19] Complete:

- Types of true aneurysms are :

a- Congenital aneurysm
b- Atheromatous aneurysm
c- Syphilitic aneurysm
d- Dissecting aneurysm
e- Mycotic aneurysm

Respiratory System

JULY 2011

[1] A non smoker 40 years female complaining of dyspnea in the last year . Chest X-ray revealed bilateral hyperinflation of the lungs .Laboratory investigations revealed alpha-1 anti-trypsin deficiency.

a- What is the most likely diagnosis?

Panacinar emphysema

b- Define the lesion.

- Permanent dilatation and enlargement initially of alveolar ducts and alveoli and later, destruction of respiratory bronchioles involving entire acini

c- Explain the cause of dyspnea in this patient.

- Expiratory airflow obstruction from loss of elastic recoil of alveoli

d- Explain the pathogenesis of this dysnea.

-In this type of emphysema, there is reduced diffusing capacity due to extensive lung destruction by the increased elastase activity due to imbalance between it and the deficient alpha 1 anti-trypsin which act as an inhibitor for elastase.

-And to maintain a normal PACO2 at rest , the body shift to hyperventilation so dypnea is produced

e- Which part of the lung is liable to be more affected? And why?

-the anterior border of the lung & more severe at the base because according to ventilation perfusion ratio, at the base there are much capillaries so more neutrophils and more elastase so more destruction

f- List other two different lesions in the lung having the same category of this lung lesion.

-irregular emphysema

-para-septal emphysema

-compensatory emphysema

-centri-lobular emphysema

g- Name other organ than the lung in this patient which must be investigated for a possible pathology

-heart

-the possible pathology : right side heart failure due to pulmonary hypertension

h- List two causes of death that may occurs in this patient.

- Massive lung collapse
-Respiratory acidosis and coma

SEP 2011

[2] A 40-year old woman had suffered from multiple attacks of necrotizing pneumonia. She now has a productive cough of large amount of purulent sputum especially on chainging position and early in the morning . the chest x-ray suggested of right lower lobe bronchi

a- What is the most likley diagnosis?

- Bronchiectasis.

b- List other suppurative lung disease.

-Bronchopneumonia -Empyema

-Lung abscess -Gangrene

c- Explain the pathogenesis of this disease.

- Necrotizing suppurative infection produce chronic bronchial wall inflammation ending in fibrosis and weakening of the wall of the bronchus
- if complete obstruction of the lumen of nearby bronchus occurs due to any cause , it will give rise to collapse of alveoli dragging with it the wall of bronchioles which are fibrosed leading to their dilatation which is permanent even if complete obstruction is removed
- these dilated bronchi will help in accumulation of secretion adding more infection and more fibrosis of the wall helping more in the pathogenesis of bronchiectais

d- Describe the microscopic appearance of this disease.

- Bronchi: dilated& filled e acute inflam. exudate "PNL & Pus"
- Epith. : necrotizing ulcer± hyperplasia or sq. metaplasia
- Wall of bronchi: acute & ch. exudate "PNL, lymph, plasma cells"
- Lung tissue: replaced by FT & ch. inflam. cells
- Musculoelastic tissue: destroyed and replaced by fibrous tissue

SEP 2013

[4]A 67-year-old man who is a long term smoker presents with a cushinoid appearance, persistent cough, fever, chest pain and hemoptysis. A chest x-ray reveals a 3.5 cm.hilar mass on the right and post obstructive pneumonia of the right lower lobe. Sputum cytology is positive for malignant cells. A transbronchial biopsy is performed.

a- What is the most likely diagnosis?

- Small cell carcinoma

b- Describe the microscopic picture of this lesion?

- Neurosecretory granules→ " ectopic hormones " are seen by E/M in malignant cells (mention the criteria of malignancy)

c- What is the cause of cushinoid appearance?

- Paraneoplastic syndrome characterized by secreting ectopic adrenocorticoid hormones by the malignant cells " secretory granules " leading to this cushinoid appearance

d- Mention other histologic structures of lung carcinoma

- Sqaumous cell carcinoma
- Adenocarcinoma " acinar , papillary , bronchioalveolar , solid mixed subtypes
- Large cell carcinoma
- Adenosquamous carcinoma

JUNE 2013

[4] A 40 year old woman had suffered from multiple attacks of pneumonia. She now has productive cough of large amount of purulent sputum specially on changing position early in the morning. The chest X-ray suggested dilatation of the right lower lobe bronchi.

a- What is the most likely diagnosis?

Bronchiectasis

b- Explain the pathogenesis of the disease?

- Necrotizing suppurative infection produce chronic bronchial wall inflammation ending in fibrosis and weakening of the wall of the bronchus
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- Lung tissue: replaced by FT & ch. inflam. Cells
- Musculoelastic tissue : destroyed and replaced by fibrous tissue

d- Explain why this patient may develop renal failure later on

- Chronic toxemia leading to amyloidosis leading to renal failure

JUNE 2013

[5] Explain increased incidence of brain abscesses in patient with suppurative lung disease?

- Due to spread of septic emboli across para vertebral plexuses of veins reaching the brain

JUNE 2014

[6] A 55 year old man had gradual increasing dysnea. He is a heavy smoker. He had a barrel-shaped chest. His chest X-ray reveled bilateral hyperinflation of lungs more in the upper lobes.

a- What is the most likely diagnosis?

Centrilobular emphysema

b- What is the pathogenesis of the disease?

- Necotin leads to chemo-attractiion and Accumulation of neutrophils and macrophages around respiratory bronchioles and by damaging lysosomes induce them to release elastase
- Oxidants in cigarette smoke inhibit the action of alpha 1 antitrypsin (functional deficiency) that is :
  - Synthesized by the liver
  - Present in serum and tissue fluids
  - Act as inhibitor of protease

  SO , the net result is imbalance between them = increasing in the elastase activity with decreased anti elastase activity in the region of terminal air spaces in the respiratory bronchioles leading to destruction of their walls

c- What are other complications of this disease?

- Cyanosis
- Pulmonary hypertension and right sided heart failure
- Polycythaemia
- Haemoptysis
- Interstitial emphysema

d- List causes of death in this disease?

- Right sided heart failure
- Respiratory acidosis and coma

SEP 2014

[7] A young male 25 year suffered suddenly from fever, dysnea , chest pain with severe cough and small amount of rusty sputum . Chest X-ray reveals consolidation of right lower lobe. Culture examination of sputum reveals gram positive cocci in pairs and chains.

a- What is the most likely diagnosis?

- Lobar pneumonia

b- Describe the pathological features in details.

1- Congestion in 1st 24hr

2- Red hepatization = consolidation

- Alveoli filled e fibrinous exudate, PNL, RBC
- Red, firm, liver like gross appearance

3- Grey hepatization:

- Alveoli filled e " fibrinous exudates + Hemosiderin produced from RBC disintegration " → grey brown app.
- Alveolar cap. → congested.
- Alveoli → thickened.
- Pleura → fibrinous pleurisy.

4- Resolution: exudates liquefied gradually by proteolytic enzymes of dead neutrophils & absorbed by lymphatics → LN hilar and bronchial.

c- If recovery does not occur, what is the possible complication?

- Abscess formation.
- Empyema (spread of infection to the pleural cavity).
- Pericarditis (spread of infection to the pericardium).
- Organization of exaudate into fibrous scar tissue.
- Bacteraemia with spread of infection to other organs producing pyemic abscesses in organs like kidney , spleen , brain , heart valves " acute bacterial endocarditis ".

SEP 2012

[8] A 2- year old child complained of fever , dysnea and cough with expectoration after an attack of whooping cough. Sputum examination reveals staph aureus organisms. Radiology revealed bilateral basal shadows.

a- What is the most likely diagnosis?

- Bronchopneumonia

b- Describe the microscopic features of this lesion?

- Lumen of alveoli & bronchi: filled e pus " suppurative exaudate '
- Epith.: destroyed
- Mucosa : ulcerated
- Wall: infiltrated with PNL, macrophages
- There is vascular hge, congestion

c- **What are the two processes involved in the pathogenesis?**

- Bacterial virulence and host resistance
  - ✓ Staph aureus is a virulent bacteria.
  - ✓ Child with whopping cough: low resistence.

  Leading to suppurative inflammation and presence of intra-alveolar inflammatory exaudate leading to patchy consolidation of lung tissue

JUNE 2012

[9] A heavy smoker 58 years old man suffers from worsening cough for several months, followed by dyspnea . He is easily fatigued with unexplained weight loss. a chest x-ray shows a 5 cm right hilar lung abscess . Sputum cytology show small malignant cells.

a- **What is the most likely diagnosis?**

- Small cell carcinoma

b- **What is the most important predisposing factors for this condition?**

- Smoking

c- **Name other lung cancer related to the same predisposing factor.**

- Squamous cell carcinoma
- Cancer larynx

d- **List three associated paraneoplastic syndrome?**

- ✓ release of parathyroid like hormona e Sq.Cell Carcinoma →hypercalcemia
- ✓ ↑ACTH → Cushing's $
- ✓ ↑ADH → urine retention &hyponatremia
- ✓ Neuromuscular syndrome : myasthenia, Peripheral Neuropathy , Poly Myositis

e- **Explain the occurrence of Horner's syndrome in cancer lung?**

- Due to " pancoast,s tumor " which is apical lung cancer in sup. pul. Sulcus
- invade cervical sympathetic plexus→ **Horner's syndrome**

Hepato-biliary System

SEP 2013

[1] A female patient 45 year old complains of attack of nausea and diarrhea and melena. Abdominal sonography reveals a benign cystic focal lesion of liver. On aspiration reveals necrotic debris with no neutrophils or pus cells.

a- **What is the possible diagnosis?**

- amebic liver abscess

b- **Can you guess the relation between colonic manifestation and hepatic lesion?**

- Complication of amoebic dysentery.
- Vegetative forms "emboli" of entamoeba histolytica carried by the portal circulation from large intestine to the liver to produce amoebic liver abscess by their liquefactive enzymes.

c- **Describe the microscopic features of this lesion?**

➢ 3 layers :

1. Innermost layer: liquefactive N. + amoebae ± RBCs (Amoeba surr. By clear halo & has spherical nucleus).
2. Next layer: few mononuclear cells + variable granulation tissue & little fibrosis.
3. Outermost layer; compressed but normal liver tissue.

d- **What are the possible complications of this lesion?**

- ➢ rupture in peritoneum→ peritonitis
- ➢ rupture in <u>stomach or Tr. Colon</u>
- ➢ if near to <u>upper</u> surface:
  - Press on lymphatic's → Rt. pleural effusion.
  - Become adherent to the diaphragm → rupture into pleura or pericardium.
  - Rupture into lung → cough contents of abscess.
- ➢ <u>Blood</u> spread → amoebic abscess in lung, spleen, rare in brain.

e- **Give 2 examples of cystic lesions of the liver.**

- Infected hydatid cyst
- Actinomycosis

JUNE 2013

[2] A 48 year old female has long history of viral liver disease. She is presenting with weakness , anorexia , loss of weight . on abdominal ultra sound a focal lesion of the liver is found.

a- What is the most likely diagnosis?

- Hpatocellular carcinoma

b- How can you confirm this diagnosis?

- Serum α fetoprotein is elevated in 80% of cases

c- List possible histopathological types.

- Clear cell type.
- Anaplastic of giant cell type.
- Sclerosing type.
- Fibrolammellar type.
- Acini & pseudoglandular type.
- Trabecular growth pattern.

d- List other four predisposing factors?

1- Steroid hormaones:

- There is link between ♀& ♂ steroids and HCC but still need confirmation.

2- Aflatoxin:

- Aflatoxin is produced by the fungus "Aspergillus Flavus" that contaminates certain stable food.
- It is experimental hepatocarcinogenic substance.

3- Cirrhosis:

- Due to e.g. alcohol or haemochramatosis

4- HBV infection:

- HBsAg +ve in high % of cases
- by integration of HBV DNA into Genome of hepatocytes

5- HCV infection

e- Lately, this patient developed dyspnea , pleural effusion , fracture of femur. What is your explanation?

- Due to metastasis to bone & lung

f- Complete :

- **Increase serum alfa feto protein ocurrs in cases of …… & ……**

1- Hepatocellular carcinoma 2- endodermal sinus tumor

SEP 2012

[3] Explain: occurrence of portal hypertension in bilharzial periportal fibrosis.

Due to development of anastomosis between the branches of hepatic artery and portal vein leads to transformation of the higher arterial pressure to the portal veins

JUNE 2012

[4] A 45- years old man had received blood transfusion. After 5 months he presented with fatigue & slight malaise. Laboratory examination reveals mild elevation of liver enzymes Hbs Ag, ANA, SMA were negative. Anti HCV was positive.

a- What is the most likely diagnosis?

- Acute viral hepatitis

b- What are the types of the degeneration that may be seen in hepatocytes? and explain their cause?

1- Ballooning degeneration :

- Swelling of the hepatocytes with indistinct cell membrane and rarified cytoplasm showing perinuclear condensation due to
  Hepatocyte injury by the virus → damage to Na K pump→ collection of Na+ inside the cell dragging water by the osmotic pressure →swelling of the cells

2- Acidophil degeneration :

Shrinkage of the hepatocytes with increased eosinophilia of the cytoplasm , pyknosis and eventual loss of the nucleus→ acidophil body " council man body

c- List two complications?

- Cirrhosis
- Hepatocellular carcinoma

d- How the activity of chronic hepatitis is evaluated?

- Modified Knodell.s scors " histopathological **activity** index " : is graded from (0-18)
  1. Portal inflammation → 0-4
  2. Periportal hepatitis " piece meal necrosis "→ 0-4
  3. Focal necrosis → 0-4
  4. Confluent nerosis → 0-6

SEP 2012

[5] A 56 year old male complains of fatigue and loss of weight for several months without any gastrointestinal symptoms. He gave a history of previous car accident and blood transfusion. on examination the patient had jaundice , firm nodular liver and mild ascites. Laboratory studies revealed elevated enzymes: raised bilirubin and decreased albumin

a- What is the most likely diagnosis? Mention etiological cause?

- post hepatic cirrhosis " due to previous viral infection ".

b- List 3 other etiological causes for this liver lesion?

- Alcohol "alcoholic cirrhosis".
- Biliary cirrhosis.
- Cholestasis.
- Drugs and toxins e.g. haemochromatosis , wilson,s diseases.

c- Explain the pathogenesis of ascites in this case?

- Partly due to portal hypertension and partly due to the accompanyling hypoproteinaemia.

d- Mention the most serious complication of this disease and the serologic test you suggest that help in the diagnosis of this complication?

- Heoatocellular carcinoma.
- Serum alpha fetoprotein is elevated in 80% of cases.

Gastro Intestinal System

MAY 2009

[1] A female 25 years old complain of recurrent colicky pain , bleeding per rectum . colonscopy reveals sessile rectal polyp measuring 2×2 cm showing velvety papillary surface

a- What is the most likely diagnosis of this polyp

- Villous adenoma

b- Give an account on different intestinal polyps (types and complications)

1- <u>neoplastic polyps :</u>

a- <u>Adenomatous polyps</u>

	Tubular	Villous	Tubulovillous
	M/C colonic adenomas	Less common	Both features
NO.	Multiple	Usually solitary	
Size	Usually < 2 cm	Large	
Shape	Pedunculated	Sessile e soft papillary surface	
MP	Tubules in FV core	Villi in FV core	
Risk	Premalignant	Higher premalign	intermediate

b- <u>Familial Adenomatous polyposis</u> = polyposis coli

- autosomal dominant
- 100 % premalignant
- No. : innumerable
- Site : colon

2- <u>Non-neoplastic</u>

- Bilharzial polyp: common in Egypt
- Hyperplastic = metaplastic: not preneoplastic
- Juvenile = retention: classed as hamartomas
- Peutz Jeugher's syndrome :
  CCC by: intestinal Polyps + mucocutaneous melanin pigmentation
- Inflammatory: e UC, Crohn's, amoebic & bacterial dysentery
- Lymphoid: Submucosal hyperplastic lymphoid tissue almostIn rectum

➢ **<u>Complications of neoplastic polys :</u>**

- Ulcer and hemorrhage
- Intussusceptions
- Malignant transformation

JUNE 2013, SEP 2008

[2] A young man suffered from retal bleeding. colonoscopic examination showed hemorrhagic colonic mucosa with ulceration. No masses were detected.

a- What is the most likely diagnosis?

- Ulcerative colitis.

b- Describe the histologic picture of the disease.

- Early cases :
  - Mucosa→ congestion, hges, edema & diffuse inflammation.
  - Superficial ulcers & crypts damage & infiltrated e PNL.
  - The hallmark→ crypt abscess : dilated, degenerated crypt filled e PNL.
- Advenced cases :
  - Epith. Cells are atrophic or show dysplastic changes
  - L.p and submucosa show chr.inflam.cells (MQ & lymphocytes).
  - No granuloma.
  - No fistula.
  - **Rare fibrosis**→ absent strictures.

c- List 5 complications of this disease.

1- Intestinal complications :
   - Toxic megacolon = dilated + functional obstruction
   - Severe bleeding in acute phase & rarely perforation
   - Epithelial dysplasia "frequent" → cancer colon
   - Inflmmatory polyps

2- Extra-intestinal complications:
   - Arthritis
   - Uveitis
   - Pyoderma gangrenosa (typical necrotic skin lesion)

d- Define the precancerous condition of this disease.

- Dysplasia: "disordered cellular proliferation with abnormality of both dofferentiation and maturation".

e- Enumerate other 3 causes of ulcers in large intestine.

- Crohn's disease.
- Amoebic dysentery.
- Bacillary dysentery.
- Adenocarcinoma of large intestine.

SEP 2013, SEP 2007

[3] A female patient 50 year old complained of epigastric pain and hematemesis. Upper gastro-intestinal endoscopy revealed a well- defined ulcer with a clean floor at the greater curvature of the stomach.

a- What is the most probable diagnosis?

- Peptic ulcer

b- Explain the pathogenesis of this ulcer?

- Imbalance between :

Mucosal defense mechanisms	Damaging forces
-Mucus: insoluble gel adherent to mucosa -Bicarbonate: neutralizes the acid -Epithelium& its regeneration: cellular barrier -Mucosal blood flow : keep integrity of mucosa -Prostaglandin→ cytoprotection	- Gastric acid & pepsin - Impaired regulation of acid secretion dt unclear causes -H. pylori "major role" • Presents in 90 – 100 % of cases of DU & 70 % of those e GU • Produce urease & protease → mucosal inflam. →mucosal injury -Ischemia & shock - Delayed gastric emptying - Duodenal gastric reflux - Antiprostaglandins "aspirin & NSAID" - Alcohol, smoking

c- What are the possible complications of this ulcer?

- Hemorhage "common" → hematemesis dt erosion of Thin vessels in ulcer base.
- Perforation: → subdiaphragmatic abscess or Generalized peritonitis.
- Cicatricial contraction: →
  - pyloric stenosis & dilated stomach.
  - Hour-glass stomach if ulcer on lesser curvature.
  - Malignant transformation "rare".
  - Penteration : : if adherent to underlying organ e.g. pancreas.

d- Enumerate 4 causes of hematemesis.

a- Local :

- Osephageal causes:
  1. Ruptured osephageal varices.
  2. Peptic ulcer of osephagus.
  3. Carcinoma of osephagus.
  4. Rupture of aortic aneurysm into osephagus.

- Gastric and duedenal causes:
  1- Acute and chronic gastritis.
  2- Gastric erosion.
  3- Peptic ulcer.
  4- Gastric carcinoma.

b- Generalized:
- Hemorhagic blood diseases e.g purpura.
- Scurvey.

JUNE 2014

[4] A 62- year old man has had anorexia , vomiting and vague abdominal pain accompanied by weight loss of 6 kg over the past 2 months , physical examination reveals supraclavicular non-tender lymphadenopathy . he becomes progressively cachertic .Upper GIT endoscopy reveals a fungating tumor mass projecting into the gastric lumen with superficial ulceration.

a- What are the predisposing factors for the development of his illness?
- Chronic atrophic gastritis with intestinal metaplasia.
- Adenoma: 40 % of gastric adenoma harbor carcinoma at time of diagnosis.
- After **gastrectomy** in the remnant stump of stomach.
- Slightly ↑ risk in persons e blood group A.

b- What is the probable histological type of this gastric tumor? Describe the microscopic pictures of its subtypes.
- Type : Adenocarcinoma.
- Subtypes :

• **Intestinal :**
- Assoc. e intestinal metaplasia and exhibits the polypoid expansile growth pattern.
- **Cells:** columnar & mucin producing, form glandular structures with papillary formation or solid sheets.

• **Diffuse (infiltrative) :**
- Poorly differentiated.
- **Cells:** single mucin producing = signet ring cells without cohesions.
- Diffuse + desmoplastic stroma (extensive fibrosis).

c- What are the methods of spread of the tumor?

1- **Direct** : to surroundings e.g pancreas , liver , transverse colon , omentum , spleen

2- **Lymphatic:**
- Lesser curvature L.Ns "**M/C**" then
- To LN along greater Curvature , porta hepatis & celiac axis L.Ns

3- **Blood**→ portal vein → liver → lungs, kidney, bones

4- **Transcoelomic** esp. to ovary → "Krukenberg tumour "

d. Discuss effects and complications of the disease.
- Nausea & abdominal pain leads to:
- Cancer cachexia with weight loss & marked anemia & anorexia
- Pyloric obstruction → vomiting
- Hge → hematemesis
- Gastric juice showing:
  - Hypochlorhydria or achlorhydria
  - Lactic acid dt fermented retained food
  - Cancer cells
  - RBCs

SEP 2014

[5] Explain: adenomatous polyp of colon is more dangerous than hyperplastic polyp.
- As adenomatous polyp has a malignant potential while hyperplastic polyp is non-neoplastic.

JUNE 2012

[6] A female patient 35 years old complained of recurrent attacks of diarrhea, fever & abdominal pain. X-ray revealed stenotic ileal segment, with skip areas. Mucosa shows cobblestone appearance and fissuring ulcers. Mesenteric lymph nodes are enlarged.

b. What is the most likely diagnosis?
- Crohn's disease

c. Explain the pathogenesis of this disease.
- Several non-confirmed factors e.g.
  - immune mediated damage to the intestine
  - T cell dysfunction
  - Several infectious agents

d. Describe the microscopic appearance of this lesion.
- All layers→ chronic 26nflame.
- Ulcers→ small, superficial, fissure + mucosal & sub mucosal edema.
- The hallmark → discrete non caseating sub mucosal granulomas.
- Fibrosis → stricture.

e. List 4 possible complications.

- Chronic diarrhea → malabsorption.
- Fibrosis → stricture.
- Fistula to surroundings"adjacent viscera, abdominal and perineal skin, U.B, vagina".
- Colonic bleeding & perforation.
- Increase risk of cancer is 3 times > normal.

SEP 2011

[7] A male patient 56 years old complains of abdominal discomfort and loss of weight. Clinical examination revealed enlarged left supraclavicular lymph node .upper GIT endoscopy reveals large ulcer at lesser curvature in the antropyloric region with thickening of gastric wall. The ulcer is 8 cm in diametèr, with raised everted edge and necrotic floor.

a- What is the most likely diagnosis?

- Carcinoma of the stomach "malignant ulcer".

b- List 4 possible risk factors?

1- Chronic atrophic gastritis with intestinal metaplasia & in pernicious anemia.
2- Adenoma: 40 % of gastric adenoma harbor carcinoma at time of diagnosis.
3- After **gastrectomy** in the remnant stump of stomach.
4- Slightly ↑ risk in persons e blood group A.

c- Describe the microscopic types of this lesion?

- Type: Adenocarcinoma.
- Subtypes :

• **Intestinal :**

➢ Assoc. e intestinal metaplasia and exhibits the polypoid expansile growth pattern.
➢ **Cells:** columnar & mucin producing, form glandular structures with papillary formation or solid sheets.

• **Diffuse (infiltrative):**

➢ Poorly differentiated.
➢ **Cells:** single mucin producing = signet ring cells without cohesions.
➢ Diffuse + desmoplastic stroma (extensive fibrosis).

d- What are the other causes of gastric ulcer?

- Ischemia & shock.
- Delayed gastric emptying.
- Duodenal gastric reflux.
- Antiprostaglandins "aspirin & NSAID".
- alcohol, smoking
- H.Pylori

e- What is the cause of enlargement of left supraclavicular lymph node and explain how this occurs?

- Retrograde lymphatic spread "Lymphatic obstruction by tumor cells may result in spread of tumor cells against the flow of lymph. This may lead to metastasis in unusual sites e.g. carcinoma of prostate, colon and stomach as we found here to supraclavicular lymph nodes"

[8] Compare between each of the following: crohn's disease and ulcerative colitis (6 differences).

	Crohn's disease = regional enteritis	Ulcerative colitis
Gross:		
-Bowel region	Ileum > colon	Colon only
-Distribution	Skip lesions	Diffuse
-Stricture	Early	Late / rare
-Dilatation	No	Yes
-Wall	Thick	Thin
-Pseudopolyps	No / slight	Marked
MP:		
-Ulcers → serositis → fistulas & sinuses	-Deep linear ulcers → marked serositis → fistulas & sinuses	-Superficial ulcers → mild or no serositis → no fistulas
-Lymphoid reaction, granulomas, fibrosis	-Marked reaction, granulomas "50 %", marked fibrosis	-Mild reaction, no granulomas, mild fibrosis
Cp:		
Fat & vitamin malabsorption	Yes	No
Comp.		
Malignant potential	Rare	Common
TTT:		
Response to surgery	Poor	Good

SEP 2012

[9] A 30 year old heavy smoker male complains of burning epigastric pain. The pain is worse at night and relieved by alkalis. Upper endoscopy reveals 2 cm mucosal defect at the antrum.

a- What is the most likely diagnosis?

- Peptic ulcer

b- List other possible sites for such a lesion?

1. 1st part of duodenum "DU
2. lower part of esophagus due to reflux
3. Zollinger Ellison syndrome ES in duodenum, stomach or jejunum
4. Meckel's diverticulum with ectopic gastric mucosa
5. Stomal ulcer " at margins of gastroenerostomy".

c- Describe the pathogenesis of this lesion?

Imbalance between:

Mucosal defense mechanisms	Damaging forces
-Mucus: insoluble gel adherent to mucosa. -Bicarbonate: neutralizes the acid. -Epithelium& its regeneration: cellular barrier. -Mucosal blood flow: keep integrity of mucosa. -Prostaglandin→ cytoprotection.	- Gastric acid & pepsin. - Impaired regulation of acid secretion dt unclear causes. -H. pylori "major role" • Presents in 90 – 100 % of cases of DU & 70 % of those e GU. • Produce urease & protease → mucosal inflam. →mucosal injury. -Ischemia & shock. - Delayed gastric emptying. - Duodenal gastric reflux. - Ant prostaglandins "aspirin & NSAID". - Alcohol, smoking.

d- List 4 complications of this lesion.

- Hemorhage" common "→hematemesis dt erosion of thin vessels in ulcer base.
- Perforation: → subdiaphragmatic abscess or Generalized peritonitis.
- Cicatricial contraction: →
  - ✓ Pyloric stenosis & dilated stomach.
  - ✓ Hour-glass stomach if ulcer on lesser curvature.
- Malignant transformation "rare".
- Penetration: if adherent to underlying organ e.g. pancreas.

JULY 2011

[10] A 40 year old man had a past history of recurrent attack of diarrhea and bleeding per rectum with a pattern of exacerbation and remission. Recently he has constipation with bleeding per rectum. Colonoscopy revealed a large ulcer with raised everted edges in sigmoid colon.

a- What is the diagnosis of this ulcer?

- Adenocarcinoma of large intestine

b- Name the predisposing diseases of such lesion?

- preexisting adenoma & ulcerative colitis"UC"

c- List the microscopic variants of such colonic ulcer?

- Adenocarcinoma
- Mucoid carcinoma
- undifferentiated carcinoma
- Signet ring cell carcinoma "poor prognosis"

d- Name the staging systems for such a lesion?

- Duke's classification :
  - ✓ Stage A: confined to wall
  - ✓ Stage B: invades precolic fat without LN
  - ✓ Stage C: as B + LN metastasis
  - ✓ Stage D:Distant metstasis

Female Genital System

MAY 2008

[1] Discuss the pathology of complete hydatiform mole (Definition, gross & microscopic picture)

- Definition :
  - **Benign** neoplasm from chorionic villi of placenta with a clinical picture of:
    - **+ ve** pregnancy test + ↑↑**serum BHCG.**
    - Uterus enlarges > normal pregnancy.
    - Vaginal bleeding in 3rd-4th month, passage of cysts.
- Gross :
  - Uterus enlarged (Wt of this mass is >200gm)
  - Uterine cavity filled 'e mass of thin walled translucent grayish white cysts "Grape-like structures".
  - No normal foetal parts.
- Microscopic picture :
  - Sheets of proliferated cyto & syncytiotrophoblasts.
  - Interior is filled 'e avascular loose myxoid stroma.
  - Cysts are dilated chrionic villi.
  - ±Cytological atypia.
- Complications :
  - 10% invasive mole.
  - 2.5% choriocarcinoma: ↑atypia &detected by early follow up of BHCG.

JUNE 2013

[2] A pregnant woman suffered from uterine bleeding. On evacuation, the uterine contents show grape like structure with thin wall, no fetal parts were found

a- What is the most likely diagnosis?

- Complete Vesicular mole.

b- Describe the histologic picture of this lesion.

- The cysts are composed of dilated chorionic villi.
- The interior is filled with avascular loose myxoid stroma.
- Trophoplastic proliferation: sheets of cyto and syncytiotrophoblasts.
- Cytologic atypia may be present.

b- Describe the microscopic picture of the lesion.

- Consists of interlacing bundles of smooth muscle fibers with dense colagen fibers and fibroblasts.
- The stroma is vascular.

c- Describe the pathological changes causing this suddenly evolving clinical presentation.

1- Red degenertion "hemorrhagic infarction":
   - Occurs in ass. With using of OCP.
   - Due to venous thrombosis or twisting of the pedicle.
   - The leiomyoma becomes soft, hemorrhagic and infracted.

2- Necrosis due to:
   - Decrease in the blood supply in the center of large leimyoma leading to torsion of the pedicle of a pedunculated tumor.

d- Mention other possible complications which might occur.

- Infection and suppuration especially in submucous type.
- Malignant change "leiomyosarcoma".
- Infertility "interfere with implantation".
- Foetal malpresentation "tumor prevent rotation of the feotus".
- U.B disorders.

e- List other causes of abnormal uterine bleeding. (MAY 2008)

- Dysfunctinal = endometrial hyperplasia.
- Complications of pregnancy:
   - Abortion.
   - Ectopic pregnancy.
   - Hydatiform mole.
   - Choriocarcinoma.
- Organic lesions:
   - Adenomyosis.
   - Carcinoma of the body and cervix.
   - Polypi.
- General causes:
   - Hypertension.
   - Blood diseases.
   - Vit.C &K deficiency.

SEP 2014

[5] A female patient 42 year old complaining of abdominal pain. sonography reveals a solid ovarian tumor . She under went surgery. The tumor was well circumscribed, firm and rubbery. Sections examined show nests ot transitional like epithelium separated by fibrous stroma.

a- What is the most likely diagnosis?

- Brenner tumors " transitional cell tumor".

b- What is the cell of origin of this tumor?

- Derived from the metaplasia of the Surface epithelium into the typical urothelial lining.

c- Enumerate other tumors having the same histogenesis.

- Serous tumor
- Mucinous tumor
- Endometrioid tumor

d- Mention the complications of ovarian tumors.

1- Torsion or twisting of the pedicle → cut of the blood supply → infarction followed by gangrene of the tumor → acute abdominal pain and hemorrhagic shock.
2- Rupture which may lead to pseudomyxoma peritonii.
3- Malignant transformation.
4- Infection that may acquired :
   - Haematogenously
   - By lymphatic spread
   - By Direct spread
5- Pressure symptoms:
   - Veins → Edema of lower limb.
   - U.B & ureters → Obstructive urinary tract troubles.
6- Sterility: if the tumor is bilateral and replacing most of the ovaries.
7- Meig's syndrome.

JUNE 2012

[6] A 35 year old female has an ovarian cystic tumor which is unilocular and measures 8 cm in diameter. It contains thick sebaceous material.

a- Which is the msot likely diagnosis of this ovarian mass?

- Mature cystic teratoma.

b- What is the cell of origin of this tumor?

- Totipotent cells " germ cells".

c- Classify this tumor.

- Mature : a- solid b- cystic "dermoid cyst".
- Mature teratoma with malignant transformation.
- Immature.

d- List 4 complication of this tumor.

1- Torsion or twisting of the pedicle → cut of the blood supply → infarction followed by gangrene of the tumor → acute abdominal pain and hemorrhagic shock.
2- Rupture which may lead to pseudomyxoma peritonii.
3- Malignant transformation.
4- Infection that may acquired :
   - Haematogenously.
   - By lymphatic spread.
   - By Direct spread.
5- Pressure symptoms :
   - Veins → edema of lower limb.
   - U.B & ureters → obstructive urinary tract troubles.

SEP 2011

[7] A 47- year old woman complained of pressure sensation , but no pain , in her pelvic region for the past 5 months . On physical examination there was a right adnexal mass . An ultrasound scan showed a 10 cm mass in the right ovary this mass was removed , it was a solid mass with cystic areas containing serous fluid exophytic papillary growths and areas of hemorrhage and necrosis

a- What is the most likely diagnosis of this ovarian lesion?

- Serous cyst adenoarcinoma.

b- Mention the cell of origin of this tumor.

- Surface epithelium tumor.

c- Describe the microscopic picture of a biopsy from this mass.

- The cyst wall is lined by cubical or sometimes columnar epithelium.
  Presence of stromal invasion & cytological atypia such as:
  - Increased nuclear cytoplasmic ratio.
  - Nuclear hyperchromatism.
  - Prominent nucleolus.
  - Increase mitotic activity.

d- List methods of spread.

- Local → near-by pelvic structures.
- Lymphatic → pelvic L.Ns.
- Haematogenous → distant organs.

MAY 2010

[8] A 60-year old infertile woman with postmenopuasal bleeding pelvis. U/S revealed: ulcerating mass 5 cm in diameter fungating within the cavity of the uterine body. Microscopically: malignant acini were seen.

a- What is the possible diagnosis of such mass?

- Carcinoma of endometrium.

b- List four risk factors which may predispose to such lesion.

1.Infertilty 2. D.M 3. Hypertension 4. Obesity

5. Prolonged estrogen stimulation as in :
   - Endometrial hyperplasia
   - Ovarian estrogen secreting tumors
   - Exogenous estrogen

c- Describe the microscopic picture of such lesion.

- Well differentiated adenocarcinoma : resemble normal endometrial glands "malignant acini" & may show:
  1- Squamous differentiation which if :
     - Benign : " adenoacanthoma "
     - Malignant : " adenosquamous carcinoma "
  2- Mucinous or secretory differentiaition

d- What is the importance of examination of the cervix in hysteretomy specimen of this patient?

- Staging of endometrial carcinoma :
  - Stage I : confined to uterus
  - Stage II : spread to cervix
  - Stage III : spread to pelvix
  - Stage IV : spread outside the true pelvis , may involve U.B or rectum

e- List other 3 lesions which may arise within the uterine cavity.

1- Leiomyosarcoma
2- Vesicular mole
3- Choricarcinoma
4- Endometrial stromal tumor :
   - Low grade stromal sarcoma " endolymphatic stromal myosis "
   - High grade stromal sarcoma

Diseases of the Breast

JULY 2011

[1] A 45-yaer old woman presents with hard breast mass measuring 5×3 cm. on clinical examination, the nipple is retracted and mass if fixed to the surrounding breast tissue. Biopsy revealed: markedly atypical, mitotically active cells arranged in sheets with minimal ductal formation. immunohistochemical staining revealed : negative staining for estrogen and progesterone receptors and positive staining for Her 2 / neu

a- What is the diagnosis

- Classic invasive ductal carcinoma

b- What is the grading of this lesion

- Grade III

c- List four different variants of this lesion

- 1- tubular carcinoma
- 2- mucinous carcinoma
- 3-medullary carcinoma
- 4- inflammatory carcinoma

d- What is the prognosis of such lesion ? and why ? (at least 2 causes)

- Very bad prognosis
- Causes :
  - negative staining for estrogen and progesterone receptors and postive staining for Her 2 / neu
  - grade III with markedly atypical, mitotically active cells arranged in sheets with minimal ductal fo nation
  - size of the tumor : 5×3 cm (> 1 cm)
  - the nipple is retracted and mass if fixed to the surrounding breast ..ssue

e- If these atypical cells extend to involve the skin of nipple and areola , what is yo ir diagnosis ?

- Lymphatic invasion " Peau d' orange "

f- List another 4 different causes for mass in the breast

- Cystosarcoma phyllodes
- Fat necros
- Fibroadenoma of the breast
- Intraductal papilloma

SEP 2011, SEP 2014

[2] List the prognostic factors of cancer breast

a- Size of the primary tumor (< 1 cm = good prognosis)
b- Lymph node status and no. of lymph nodes involved
c- Stage of the disease
d- Histologic grade of the tumor
e- Histologic type
f- Estrogen and progesterone receptors (good prognosis if positive)
g- Over expression of HeR 2/neu = bad prognosis

SEP 2012

[3] A 65 year old woman complains of itching and scales with eczema-like change of the nipple in the right breast. On examination , a 1 cm palpable irregular hard mass is felt in underlying breast tissue

a- What is the most likely diagnosis of this skn lesion
- Paget.s disease of the nipple

b- Describe the microscopic findings of a biobsy from this skin lesion
- The duct cells appear as large , pale , vacuolated cells located within the overlying keratinizing squamous epithelium

c- What is the expected diagnosis of the underlying mass
- In situ or invasive duct carcinoma

d- What is the most common location of this skin lesion other that the breast and what is the difference between both sites ? (JUNE 2014)
- Vulva " labia majora "
- The difference is :

Paget.s disease of the breast is always 100% associated with underlying or adjacent ductal carcinoma while vulvular lesions are most confined to the epidermis and only become invasive in persistant untreated cases

JUNE 2013, SEP 2014

[4] Explain pathogenesis of peau d'orange of the skin in patients with breast carcinoma.

Infiltration and obstruction of dermal lymphatics by cancer cells

SEP 2014

[5] Classify breast carcinoma

1- Ductal carcinoma (90%)
   a- Duct carcinoma in situ
      - Papillary
      - Solid
      - Comedo
   b- Invasive duct carcinoma
      - Classic
      - Tubular
      - Mucinous
      - Medullary
      - Inflammatory
2- Lobular carcinoma
   a- Lobular carcinoma in situ
   b- Invasive lobular carcinoma

Male genital system

SEP 2008

[1] Give an account on classical seminoma (Gross and Microscopic).

- **Gross :**
  - Site: replacing all or parts of testis.
  - Shape: well circumscribed, spherical.
  - Consistency: solid firm.
  - Cut section: grayish white to pink & homogenous.

- **Microscopic :**
  - Masses of malignant **cells.**
  - Separated by thin vascular **CT.**
  - The cells are large e abundant.
  - Clear or pale **cytoplasm** & Large central ↑**nuclei.**
  - Stroma is infiltrated by Lymphocytes& epitheloid Granuloma resembling Sarcoidosis (favorable prognostic features).

- **Prognosis: "very radiosensitive "**
  - 95 % of patients e paraaortic LN cured by radiotherapy after Orchidectomy.
  - Also lung metastasis have good prognosis after radiotherapy.

MAY 2009

[2] A male 33 years old complain of testicular swelling . Clinically , there is a well- circumscribed spherical mass replacing most of the testicular tissue . Orchodectomy was done and the patient respond to radiotherapy

a- What is the most likely diagnosis of this swelling?
- Classical Seminoma

b- Under what category is this lesion classified
- Germ cell tumors

- **Gross:**
  - **Size:** enlarged prostate &↑in weight (N: 14gm).
  - **Site:** mainly the 2 lateral lobes & the middle lobe " behind the urethra".
  - **Consistency:** firm.
  - **C/s:** whitish , nodular ± cysts containing milk secretion , infarcts , abscesses.
- **MP:**
  - Proliferation of both prostatic acini and fibromuscular stroma.
  - The acini are variable in size , some are cystically dilated.
  - They are lined by multiple layers of hyperplastic columner epithelial cells with intraluminal micropapillary projections.
  - There are intraluminal secretions and corpora amylacea.
- **Effects and Complications :**
  - **Urethra :**
    - ✓ Compression → difficult micturition " dysuria".
    - ✓ Congestion → acute retention or hematuria.
  - **UB :**
    - ✓ Incontinence dt stretching of sphincter by middle lobe.
    - ✓ Residual urine: behind the enlarged middle lobe.
    - ✓ Cystitis & stone: dt urine accumulation.
    - ✓ Dilatation & hypertrophy.
  - **Ureters & kidney**
    - ✓ Bilateral hydroureter & hydronephrosis dt obstruction.
    - ✓ Pyelonephritis & pyonephrosis dt ascending infection.
    - ✓ Chronic RF dt hydronephrosis.

Endocrinal System

MAY 2008

[1] Give an account on simple nodular goiter.

	Simple (non-toxic) goiter			
Incidence	**Female**: male = 8:1. Peak in **young** adults.			
Etiology	1. **Endemic:**↑in areas 'e iodine ↓↓→↓production of thyroid hormone→ compensatory ↑TSH →hyperplasia& hypertrophy of the gland. 2. **Sporadic:** less common			
Pathology	Passes into 3 stages:	**Diffuse parenchymatous**	**Diffuse colloid**	**Simple multinodular**
	Pathology.	Early dt↓**iodine intake**.	**Adequate iodine**.	After **repeated cycles** of hyperplasia & involution.
	Gross.	**Diffuse** enlarged	**Diffuse** enlarged	**Nodular** enlarged,
	C/S.	-	Brown **cystic** gelatinous	**firm** Multiple **nodules** separated by FT septae. ± Areas of hge, Necrosis , cystic degeneration, calcif.
	MP.	**Hyperplasia** of Epith. Of acini 'e↓colloid.	acini distended 'e **colloid**& lined by flattened epith.	Multiple **nodules** of acini of variable size, shape ±colloid + Acini separated by ft 'e↑lymphocytes.
Manifestation	1. **Pressure effects:** • On trachea→ dyspnea. • On esophagus→ dysphagia. • On Recurrent Laryngeal Nerve → hoarseness of voice. • Retrosternal space→ **retrosternal goiter → mediastinal $** 2. **Toxicity:**2ry toxic goiter			

SEP 2013

[2] Give reason: Dysphagia and hoarseness of voice may occur in cases of simple goiter.

Due to pressure effects on

- Esophagus → dysphagia.
- Recurrent laryngeal nerve → hoarseness of voice.

JUNE 2013, SEP 2012

[3] The nuclear features of papillary carcinoma of thyroid are ..... & .....

- Ground Glass nuclei="orphan Anne eye" =hypo chromatic empty nuclei devoid of nucleoli
- Nuclear grooves.
- Eosinophilic intranuclear inclusions.

MAY 2009

[4] Discuss pathology of Graves disease "primary toxic goiter" (pathogenesis , clinical manifestation , gross and microscopic picture).

	Papillary carcinoma of thyroid
Incidence	• Most common cancer • ↑in Females in 3rd - 5th decade.
Gross	Vary in size • < 1cm = micro carcinoma. • Large tumors show cysts, Hge, calcificated.
MP	• Cells arranged in layers around fibro vascular core ± follicular variant • **Psammoma bodies** "common" • Ccc nuclear features: • **Ground Glass nuclei="orphan Anne eye"** =hypo chromatic empty nuclei devoid of nucleoli • Nuclear grooves. • Eosinophilic intranuclear inclusions.
Spread	1. **Local:** infiltrates surrounding. 2. **Lymphatic:** common &early 3. **Blood:** rare & late.

MAY 2010

[5] Give an account on the pathology of papillary carcinoma of thyroid.

	1ry "Grave's ds" =exophthalmic goiter
Incidence	Common, ↑in middle aged females.
Cause	• Unknown cause. • Usually follow psychic shock. • Recently, it is autoimmune ds 'e ↑production of LATS.
Gross	Diffuse enlarge, firm.
C/S.	Red vascular like meat
MP.	• ↑↑Epith. Hyperplasia (columnar cells in >1 layer e papillae). • ↓Colloid. • Stroma: - ↑ Vascularity& - ↑ Lymphocytic infiltration.
Manifestations	1. ↑BMR. 2. CNS : nervousness, irritability, tremors& insomnia 3. Eye: Exophthalmos dt edema &↑orbital fat dt↑activity of ant. pituitary secreting "exophthalmos-producing substance" "EPS" 4. CVS: tachycardia, ↑Bp, HF. 5. L.N: generalized hyperplasia of L.Ns, tonsils & spleen. 6. Skin: ↑sweating +wet skin.

JUNE 2014

[6] Give reason: Papillary carcinoma of thyroid has better prognosis than follicular carcinoma.

- As papillary carcinoma has late and rare spread by blood but follicular carcinoma spread early by blood to lungs and bones and also capsular or vascular invasion are mandatory to confirm the diagnosis

SEP 2014

[7] Explain: Medullary carcinoma is different from all other tumors of thyroid.

- As it Represent neuroendocrine neoplasm originating from parafollicular C cells
- Distinctive features include secretion of calcitonin in most cases and amyloid stroma in many cases
- Some cases show carcinoid like symptoms i.e excessive flushing and diarrhea.

Lymph nodes and Spleen

MAY 2007, MAY 2010

[1] A middle – aged person suffers from painless non-tender enlarged lymph nodes , mild intermittent fever , progressive anemia and itching .

a- What is the diagnosis of this case?

- Hodgkin's lymphoma

b- Discuss the pathological features (gross and microscopic)?

Gross

- First: LN are enlarged, **discrete, firm (rubbery).**
- Later: **Adherent** due to invasion of the capsule.
- Cut surface :homogenous **grayish pink.**

MP

CCC by presence of :

- Malignant giant cells (**Reed-Sternberg Cells**).= **RSC**
- Background of normal **lymphocytes** & heterogeneous benign **infl. cells.**

Classical RSC	Non-specific variants of RSC			
	Mononuclear variant	**Popcorn variant (lymphocytic predominance)**	**Pleomorphic variant**	**Lacunar variant**
size : Large malignant cell **cytoplasm :** abundant weakly acidophilic or amphophilic	**Large** cell	Scanty **cytoplasm**		May retract & cell appears to lie in clear space **"lacuna or lake"**
Nucleus : Bilobed or polylobed so appears→binucleated or multinucleated. **Nucleolus :** large acidophilic central **nucleolus = "owl eye" app. ="mirror image"**	Single nucleus. large Esinophilic nucleolus May be seen in any subtype of Hodgkin.	Nucleus puffy, multi-lobed Punctuate nucleoli.	Single nucleus, irregular, **multi-lobed**. Variable Nucleoli	Single **multi-lobed** nucleus. **Nucleolus** is relatively small.

c- Describe the microscopic subtypes of such lesion.

➢ According to:

- Frequency& types of RSC.
- Predominant background cell type
- Occurrence of fibrosis.

Lymphocytic predominant type	Mixed cellularity type	Lymphocytic depletion type		Nodular sclerosis type
• **Classical RSC** are rare 'e relative frequency of popcorn variant. • **Background;** Normal lymphocytes in diffuse pattern replacing normal architecture.	• **↑Classical RSC.** • **Background;** diffuse, variable, **mixed infl.cells** (eosinophils, plasma cells, histiocytes & moderate No of **lymphocytes**)→ mixed cellularity	• **Classical RSC** are rare 'e pleomorphic variant. • **Background;** few **normal lymphocytes +inf.cells.** • The infiltrate is diffuse+**loose F.T** • It present in 2 morphological subtypes:		**Classical RSC** are v.rare 'e ↑No. of lacunar variant. **Back ground;** variable**normal lymphocytes +Mixed infl.cells.** Divided into nodules by **dense F.T** **The best favorable prognosis.**
		Diffuse fibrosis	**Reticular subtype**	
		Predominance of fibrosis 'e hypocellularity.	more cells & less fibrosis	

d- List other four different causes of lymph node enlargement.

1- **Acute :**
   - typhoid fever.**infection**
   - bubonic plague.
   - post –vaccinial.
   - infetious mononucleosis."glandular fever"

2- **Chronic :**
   - Cat scratch disease
   - Sarcoidosis
   - TB
   - AIDS
   - Lymphoma

JUNE 2014

[2] Compare between lymphocytic predominant and lymphycytic depletion type of Hodgkin's lymphoma?

Lymphocytic predominant type	Lymphocytic depletion type
• Classical RSC are rare 'e relative frequency of popcorn variant. • scanty cytoplasm • Puffy , multilobed'e punctuate nucleoli • Background; normal lymphocytes in diffuse pattern replacing normal architecture.	1. Classical RSC are rare 'e pleomorphic variant. • Single nucleus, irregular, multilobed. • Nucleoli are variable in size. 2. Background; Few normal lymphocytes +inf.cells. • The infiltrate is diffuse+looseF.T • It present in 2 morphological subtypes:

Diffuse fibrosis	Reticular subtype
predominance of fibrosis 'e hypocellularity.	more cells & less fibrosis

[3] Causes of spleen enlargement are ……, ……, ……

1- Infections :
- Typhoid /TB /Toxoplasmosis.
- Infectious mononucleosis.
- Malaria
- $.
- Bilharziasis.

2- Congestive state :
- Cirrhosis
- Rt. Side heart failure

3- Staorage diseases :
- Gaucher's ds.

4- Miscellaneous:
- Amyloidosis
- Primary neoplasm and cysts
- 2ry neoplasms

JUNE 2013

[4] …… is a low grade non hodgkin.s lymphoma

1- Small lymphocytic lymphoma.
2- lymphocyticplasmacytoid lymphoma.
3- Follicular lymphoma:
- Follicular small cleaved cell lymphoma.
- Follicular mixed small cleaved & large cell lymphoma.

JUNE 2012

[5] Give an account on variants oh hodgkin's reed sternberg cells (name and relation to subtypes of hodgkin's lymphoma).

Classical RSC	Non-specific variants of RSC			
	Mononuclear variant:	Popcorn variant "lymphocytic predominane"	Pleomorphic variant "lymphocytic depletion"	Lacunar variant "nodular sclerosis"
• Size : Large malignant cell	Large cell			
• Cytoplasm : abundant weakly acidophilic or amphophilic		Scanty cytoplasm		Cytoplasm may retract & cell appears to lie in clear space"lacuna or lake"
• Nucleus : Bilobed or polylobed so appears→binucleated or multinucleated.	Singlenucleus.	puffy, multilobed	Single nucleus, irregular, multilobed.	Single multilobed nucleus.
• Nucleolus : large acidophilic central = "owl eye" app. ="mirror image"	Large esinophilic nucleolus May be seen in any subtype of Hodgkin.	Punctuate nucleoli.	Nucleoli are variable in size.	Nucleolus is relatively small.

SEP 2012

[6] Give an account on starry-sky apperance in burkitt's lymphoma.

The cell features:

- It is due to Phagocytosis of nuclear debris "from rapid division& cell necrosis" by MC →starry sky appearance.

SEP 2013

[7] Causes of chromic lymph node enlargement are …… , …… , ……

- T.B , sarcoidosis , AIDS
- Lymphomas , metastatic tumors
- Chronic leukemia
- Cat scratch disease

Diseases of Bone

JULY 2011 - 2013

[1] Explain the possible causes in : septic arthritis is a rare complication of acute haematogenous osteomyelitis.

- Epiphysis prevents spread from metaphysis.
- The firmly attached periosteum to epiphyseal margins.
- Spread to nearby joint may occurs in : -
  - Intraarticular metaphysic e.g. hip joint.
  - Long tendon passing through the joint e.g. shoulder.

[2] Diagnostic microscopic findings in osteosarcoma is …….

- Fine lace like pink matrix.

SEP 2013

[3] Explain the possible causes in: sequestrum formation in haematogenous osteomyelitis.

- Occlusion of both periosteal and endosteal vessels that lead to necrosis of some or all of the diaphysis.

[4] Sunrays appearance is a characteristic radiological feature of …… while onion skin appearnace is a characteristic radiological feature of ……

- Osteosarcoma.
- Ewing's sarcoma.

JUNE 2012, 2014

[5] Compare between: osteoblastoma and osteoid osteoma (Bone affected, Site, Size)

	Osteoid Osteoma	Benign Osteoblstoma
Size	1.5 in diameter	2 cm in diameter
Affected bones	Any but ↑at femur & tibia	All over the skeleton, the vertebrae being their commonest location
Site	Cortex of metaphysis	Medulla of metaphysic

SEP 2014

[6] Complications of chronic osteomyelitis are ……

- Secondary amyloidosis.
- Squamous cell carinoma of the sinus tract.

Diseases of the joints

SEP 2011

[1] Compare between: rheumatoid arthritis and osteoarthritis (3 differences).

	Osteoarthritis	Rheumatoid arthritis
Def	Degenerative non inflammatory dt unknown cause	Chronic systemic inflammatory disorder
Age	Old age but in young by trauma & obesity	No age is immune, but ↑in 3rd -4th decade
Site	Large joints as hip, knee & vertebrae	Small joints of hand & feet > larger joints
Gross	Gradual progressive erosion of articular cartilage: •Granular articular cartilage •Sloughed cartilage •Bone eburnation: polished ivory app, dt friction •Osteophytes: mushroom shape cartilaginous Appendices then ossify	As regard joints: Gross: thick, edematous synovium + Villous folds matted by fibrin
Mp	- Chondrcyte: proliferate → clones - Synovium: congested, fibrotic ± chronic inflam. Cells	The villous fold: •lined by proliferated synoviocytes •Stroma: dense perivascular inflam. (lymphocytes, plasma cells, MCs) •Rice bodies: fibrin on margin of villi If progress: •Pannus: inflamed granulation tissue creeps Over the cartilage → erosions →Fibrous Ankylosis → bony ankylosis → deformity •Pannus extend to periarticular tissue → Joint instability •Muscle atrophy esp. extensors of fingers leads to flexion deformity

MAY 2009

[2] A male 35 years old complains of swelling in the area of right knee for more than one month. Radiological, there is a lytic lesion with "soap and bubbble appearance"

a- What is your diagnosis?

- Giant cell tumor

b- Describe the pathological feature of this lesion (sites , gross , microscopic & grading system).

	Giant cell tumour = osteoclastoma
Age	20 – 40 y
Affected Bone	Any but ↑at lower radius, upper humerus Lower femur, upper tibia
Site	Epiphysio-metaphyseal "epiphyseal line"
Gross:	Size -expand the bone & thinning of cortex "egg shell crackling sensation" Consistency -soft & fleshy + cyst formation Color -dark brown
Radio	*Tumour → bone expansion&thin cortex *Radiolucent e thin sclerotic lines ⇩ Soap bubble appearance
MP	• Cells: small stromal "spindle or oval" + Multinucleated giant cells "uniform cells, Up to 100 nuclei & abundant esinophilic cytoplasm" • Grade: 1 = Benign = the majority: ↑giant cells + ↓stromal cell 2 = Locally aggressive = potentially low grade malign. ↑stromal cell e marked atypia & moderate mitosis 3 = Malignant: stromal cells e ↑atypia &↑mitosis N.b : this grading is not of value Because metastasis can occur inspite of benign MP

Newous System

MAY 2008

[1] Write on the pathological features of acute pyogenic meningitis (gross and microscopic).

✓ **Gross**
- Subarachnoid space is filled with pus &more in basal, frontal & parietal areas.
- Congested pia, arachnoid & choroid plexus
- Mild dilated ventricles

✓ **Microscopic**
- Subarachnoid space is filled e fibrin, PNL, pus cells & histiocytes
- Dilated congested meningeal vessels

SEP 2008

[2] Discuss the pathology of intracranial aneurysms (Types and complications).

✓ **Types**

	Congenital = Berry	**Mycotic**	**Atherosclerotic**
Cause	congenital	SABE, PAN	Atherosclerosis
Site	Around circle of Willis	Branches of MCA	Internal carotid & basilar a.
Shape	Multiple saccular		Fusiform

✓ **Compilcations**
- ↑Intracranial tension → dysfunction & hydrocephalus.
- Rupture → subarachnoid hge = SAH.
- Thrombosis & calcification.

SEP 2007

[3] Discuss the pathology of meningioma.

- Grade : usually benign
- Age : adult
- Site : From covering endothelium of arachnoid (meningothelial cells) & attached to dura, embedded in brain (no invasion)
- Effects : mainly increase in intra-cranial tension
- Gross : Well encapsulated, grayish white e whorly cut surface
- MP : **Histologic patterns** with variable Prognosis:

- Syncytial: cluster cells in Groups e out cell membranes
- Fibroblastic: elongated cells +↑↑collagen e whorly app.
- Transitional: syncytial + fibroblastic.
- Psammomatous: ↑psammoma bodies.
- Papillary: pleomorphic cells around FV core (worse prognosis).
- Malignant meningioma: Unusual.
- Sarcoma: uncommon.

JUNE 2013

[4] Compare astrocytoma grade II and glioblastoma multiformans.

	Grade 2	Grade 4 = Glioblastoma Multiforme =the most malign.
Gross	Grey white, poorly defined & infiltrative	Firm white areas + softer yellow foci of necrosis , cystic change and hge (variegated appearance)
MP	Hypercellular + minimal nuclear pleomorphism	↑ Nuclear density of ↑ anaplastic cells at periphery of necrotic areas "pseudopalisading" There are tumor giant cells and mitotic figures

SEP 2013

[5] Compare CSF findings in suppurative meningitis and tuberculosis meningitis.

	Suppurative meningitis	Tuberculosis meningitis
CSF findings	- ↑Protein content - ↑ PNL & organisms - ↓Sugar content - Turbid &↑amount	-↑Protein → cob web coagulum of fibrin on standing - Cells are mainly lymphocytes - ↓Chlorides

JUNE 2014

[6] List the complications of acute suppurative meningitis.

- **General:** Septicemia →
  - Infective endocarditis
  - Acute adrenal insuff. "Waterhouse – Freidrichsen syndrome "
- **Local:**
  - Thrombosis of blood vessels
  - Encephalitis
  - ↑ICT dt fibrosis → obstruct foramina of Lushcka & Magendi → hydrocephalus in children & damage of cranial nerves 3, 4, 6 → ptosis, diplopia, squint

General

SECTION 1 | GIVE AN ACCOUNT ON

1) TB Lymphadenitis

MAY 2006, SEP 2008, MIDYEAR 2007

	Early	Late = Caseating
Gross	- Enlarged. - Firm. - Discrete. - Cut surface: grayish white.	- More enlarged. - Soft. - Matted (per adenitis). - Cut surface: cheesy yellowish opaque.
MP	• Reserved nodal architecture • Multiple small tubercles: - Epitheloid - Few langhan's - Little or no caseation.	• Lost architecture by caseous necrosis • Multiple small tubercles at periphery

- **Effects of TB Lymphadenitis:**
  - Cervical LN
    - Cold abscess→ Sinuses discharging caseation
    - spread→ erode a bronchus
    - Scrofuloderma: inflam. around sinuses → TB bronchopneumonia
    - Scar due to healing of some sinuses
  - Mediastinal LN: Mediastinal $
  - Mesenteric LN: Rupture → TB peritonitis
  - ➢ Any of the 3 group may lead to: Miliary TB, 2ry Amyloidosis, Dystrophic calcification.

2) Membranous inflammation

	Membranous inflammation
Ccc by	Pseudomembrane
Sites	- Diphtheria - Bacillary dysentery
Pathogenesis	Bacteria on mucosa → Exotoxin → patchy necrosis: * To bl. → toxemia. * To SubMucosa → acute inflam. e ↑fibrin exudate + necrotic mucosa = **pseudo membrane**.

Gross	**Pseudo membrane:** Dirty grayish white loosely attached → easily detached → bleeding ulcer so called "False or pseudomemb."
MP	**Pseudo membrane:** - Causative organism - Necrotic mucosa - Fibrin - PNL - Some RBCs

3) Complications of wound healing.

MAY 2008, 2014

1- **Ulcer:** Area not covered with epithelium dt large wound.
- May require skin graft.

2- **Keloid:** Fibrous tissue in dermis projecting over surface & covered by stretched epidermis.
- Due to ↑granulation as in burn healing dt unknown cause.
- Recur after surgical removal.
- ↑in neck &↑in Negro races.

3- **Exuberant gran. = proud flesh:** ↑gran. above level of skin but corrected by cauterization or surgery.

4- **Sinus & fistula:**
- If FB or inf. → gran. → Tract.
- If the tract has a blind end → sinus.
- If the tract opens on 2 surfaces → fistula.

5- **Weak scar:** in ant. abdominal wall → incisional hernia.

6- **Cicatrisation:** ↓size of scar as in burns → deformity.

7- **Calcification**&ossification of scar.

8- **Epidermoid Cyst:** dt implanted epidermal cells → cyst filled e keratin.

9- **Malignant Change:** SCC is rare.

4) Healing by secondary intention.

SEP2007

	2ry union = 2nd intention
In	Septic, abscess, infarction
Tissue Loss	Marked
Gap	Wide
FB & inf.	Present
Granulation	Abundant
Contraction	Present
Ends by	Large white irregular contracted avascular weak scar lacking hair follicles & sebaceous gl.
Complications	More common " the most important is suppuration"

5) Pyaemia

MAY- 2002, 2007- MIDYEAR2012

	Pyaemia
Def	Multiple small **abscesses** in organs dt septic emboli.
From	Septic focus→ Septic thrombophlebitis. →Fragmentation of thrombus → septic emboli → Pyemic abscesses- CCC by: Number: Multiple. Site: usually at periphery. Size: small , nearly the same. Surr. by: congestion "no fibrosis dt fatal condition".
Effects	**2 Types of pyaemia**

	Systemic	**Portal**
carried by	Systemic circ.	Portal circ.
As in	1-Osteomyelitis 2-Puerperal sepsis 3-Otitis media 4-Supp. lung dis. 5- ABE	- Appendicitis - Infected piles - Supp. GB = cholecystitis & Large intestine
To	- 1, 2, 3 to lung- - 4, 5 to any organs including liver	Liver

6) Epithelial-metaplsia

(MIDYEAR 2006, 2012 SEP2006, 2007, 2008)

➢ Def.:
- Replacement of one **normal adult cell** type by a **different fully differentiated adult cell** type.
- This change is of the **same category** (epith→epith) = doesn't cross histogenetic boundaries
- Usually dt **ch. irritation**.

➢ It is a **reversible** mech.

➢ Examples:

a- C.T Metaplasia:
- **Osseous:**
  - Dystrophic calcification in muscles: myositis- ossificans
- **Myeloid:**
  - Extra- medullary hemopoiesis "in liver & spleen" in case of BM myelofibrosis

b- Epith. metaplasia :

- **Squamous:**
  - From pseudo st. col. Ciliated: as In bronchiectasis, ch. Bronchitis
  - From col. Epith. In chronic cervicitis
  - From tr. Epith. In ch. cystitis & Bilharziasis
- **Columnar** :
  - Apocrine in fibrocystic ds. Of breast
  - Intestinal met. In chronic atrophic gastritis.

7) Pathological calcification

MAY 2007

➢ **Def:**

Calcium deposition in abnormal site "other than bone & teeth".

➢ **Types:**

	Dystrophic "the M/C"	Metastatic
Ca+2 level	Normal "9 – 11 mg %"	Hypercalcemia
Ca+2 metabolism	Normal	Deranged
Reversibility	Generally irreversible	Reversible upon correction of metabolic disorder
Mechanism Or causes	Necrosis or degeneration→enzymes ↓ Breakdown of organic phosphates &alteration of PH→ calcium deposition	a- Excessive mobilization of calcium from bone : e.g, • Hyperparathyroidism. • Destructive bone disease such as multiple myeloma. b- ↑ Ca absorption from intestine as: • Hypervitaminosis D. • Sarcoidosis. • "Milk- alkali syndrome".
Sites	➢ Dead tissue: - caseous necrosis - fat necrosis - dead parasites "bilharzial ova & hydatid cyst" ➢ Degenerated tissues: 1. Fibrosis as: -Fibrosed cardiac valves -Atheroma -Old scar -Wall of chronic abscess 2. Tumors as: - Leimyoma & meningioma	- Alveolar walls - Arterial wall -Fundal gland of stomach. - Kidney stones & around the tubules

8) Primary pulmonary complex.

(MAY 2008)

- Def :
  - Primary tuberculous focus at the site of infection + the tuberculous lymphangitis + tuberculous lymphadenitis
- Types :
  - a- Primary cervical complex :
    - TB of the tonsil
    - Tuberculous cervical lymphangitis
    - Tuberculous cervical lymphadenitis
  - b- Primary pulmonary complex :
    - 1ry TB of the lung " Ghon's focus "
    - Tuberculous pulmonary lymphangitis
    - Tuberculous lymphadenitis of the hilar L.Ns
  - c- Primary intestinal complex :
    - 1ry TB of intestine " 1ry tuberculous enteritis "
    - Tuberculous mesenteric lymphadenitis " tabes mesentrica "
    - Tuberculous mesenteric lymphangitis
- Sometimes the three groups are affected at the same time i.e "Primary glandular complex"

9) A- male patient is diabetic for 10years , 4 days ago he complained of pain in the back of his neck .On examination on the back of the neck it was red ,hot , swollen and there were multiple openings discharging pus

MIDYEAR 2012

a- What is your diagnosis? Specify its type?

-furuncle = boil

-localized suppurative. acute inflammation

b-what is the pathological term- of this opening ? define it?

- sinus:- abnormal tract lined by septic granulation tissue connecting a cavity to outside "1 blind end"

10) Define "Organ tropism" & mention 3 examples

MIDYEAR 2015

- Def: The arrest of tumor emboli in a specific organ is related to the expression of adhesion molecules or chemokines by the tumor cells whose receptors are expressed on the endothelium of target organ.
- Examples :
  1- Cancer breast , thyroid , kidney , prostate , lung → bone metastasis
  2- Cancer bronchus → adrenal , liver , brain metastasis
  3- Seminoma → lung metastasis
  4- Sarcoma → lung , liver , bone metastasis

SECTION 2 | COMPARE BETWEEN

1) Fistula and sinus

Sinus	Fistula
Abnormal tract lined by septic granulation tissue connecting a cavity to outside "1 blind end".	Abnormal tract lined by septic granulation tissue connecting 2 cavities or hollow organ & surface "2 opened ends".

2) Amyloid associated protein and amyloid light chain protein

JUNE 2012

AL = amyloid light chain	AA = amyloid associated
Ig light chain "mainly lambda λ"	Non Ig protein synthesized- by- the- liver
From plasma cells & assoc. e multiple myeloma	↑in ch. inflam. "e.g TB - Ch.osteomyelitis
→1ry amyloidosis	→2ry amyloidosis

3) Primary and secondary amyloidosis

MIDYEAR- 2012

	Primary	Secondary
Site	Heart, GIT, tongue, skin & nerves	Kidney, liver, spleen, LN
Cause	Unknown assoc. e multiple myeloma	Ch. inflam. as: - Chronic suppurative lung disease. - Granuloma: TB, - Rheumatoid arthritis
	AL = amyloid light chain	AA = amyloid associated

4) Hyperplasia and hypertrophy (definition and two examples).

JUNE- 2012

	Hyperplasia	Hypertrophy
Def	↑Size of a tissue dt↑- number of component cells (liable & stable cells)	↑ size of a tissue dt ↑size of individual cells (in tissue formed of permanent cells)
2 Ex	1- hyperplasia in mammary tissue during lactation 2- Endometrial. Hyperplasia dt unopposed estrogen near menopause 3- LN hyperplasia in inflammation 4- Cystic mammary hyperplasia (fibrocystic disease of the breast)	1- Lt.Ventricle-in-Hypertension 2-One kidney in removal of the other. 3-Urinary- Bladder wall in neck obstruction

5) Benign and malignant tumor

SEP2004

		Benign	Malignant
Mode of growth		Expansion	Infiltration
Rate of growth		Slow	Rapid
Grossly	Size	Small	Large
	Capsule	Encapsulated	Non capsulated
	c/s	No ulcer, hge, necrosis	Ulcer, hge, necrosis.
	Vascularity	Less marked	Marked
MP	Differentiation	Well differentiated	Poorly differentiated "not resemble tissue of origin"
	Criteria of malignancy	Absent	Present: Large pleomorphic cells variable in size & shape NC: large pleomorphic hyper chromatic + clumped chromatin + ↑N/C ratio + prominent nucleoli + ↑mitotic activity
Prognosis	Recurrence	No	Usually recur
	Metastasis	No	+ve to distant organs
Effect		Rarely dangerous except in CNS, airway obst, IO	Usually fatal
DNA		Diploid (46)	aneuploid = irregular ↑DNA

6) Carcinoma and sarcoma

MIDYEAR2007- SEP2007- SEP2002- JUNE2013

	Sarcoma	Carcinoma
Origin	Mesenchymal malignancy	Epith. Malignancy
Age	↑ in young adult	↑ in > 45 y
Growth Rate	Rapid	Less rapid
Growth Mode	More by expansion	More by infiltration
Gross: Size	Bulky	Less bulky
Consistency	Soft or fleshy	Firm or hard
Vascularity	Very vascular	Less vascular
C/S	Homogenous grayish	Grayish white
N. & He	Common	Less common
MP	↑Cellular in sheets e ↓stroma	Masses or acini + FT stroma
Spread	Early by blood	Early by lymphatic
Prognosis	Worse	relatively better

7) Locally malignant tumor and Carcinoma in situ.

MAY 2006, SEP2007, 2013

	locally malignant tumor	Carcinoma in situ
Def-	Infiltrate surrounding but no distant metastasis. "no lymphatic or bl. spread"	Pre- invasive carcinoma but no basement memb. Infiltration.
Example	- Craniopharyngioma. - BCC "Basal Cell Carcinoma". - Adamantinoma "ameloblastoma". - Giant cell tumor = osteoclastoma grade II	Cervix ,UB, Bronchus , breast.

8) Wet and Dry gangrene.

SEPT 2008

	Dry gangrene	Wet gangrene
Spread	Slow	Rapid
Toxemia	Minimal	Marked
Offensive odor	Slight	Very offensive
Putrefaction	Minimal due to dryness and presence of very little blood supply →↓bacterial growth	Marked → death due to stuffing of organ with blood
G. condition	Not bad	Very bad
Prognosis	Not bad	Bad
Line of demarcation	Present at the junction between healthy and gangrenous part	Absent / not well formed
Sites	↓tissue fluid & dry by evaporation as Lower Limb	↑tissue fluid & not exposed to dryness As LL in special cases, intestine, lung
Gross	Dry, shriveled, mummified & black	Swollen, edematous, blebs, black, rotten and ulceration of stretched skin
Mechanism	Arterial occlusion	More commonly venous obstruction

9) Haemosiderosis and Haemochromatosis.

MIDYEAR 2012

	Haemosiderosis	Haemochromatosis
Def	Presence of hemosiderin in the mononuclear phagocytes only	Progressive and more extensive accumulation of hemosiderin in mononuclear phagocytes and parenchymal cells throughout the body
Sites	Liver , BM, spleen ,L.Ns	Liver , pancreas , heart, endocrine organs
	Without tissue injury	With tissue injury
Types	- **Localized :** e.g. the changing colors of a bruise or a black eye , brown induration of flung in CVC. - **Generalized (systemic):** e.g. chronic hemolytic anemia , excessive dietary intake of iron "Bantu's disease "	- **Primary "idiopathic" :** e.g Bronze diabetes skin pigmentation + pancreas damage + liver cirrhosis - **Secondary "acquired " :** e.g. long term systemic hemosiderosis

10) Microscopic appearance of sarcoid and TB granuloma

SEPT 2012

	Sarcoid granuloma	TB granuloma
MP	Small, well circumscribed granuloma with: - Fibrosis - Epitheloid cells & langhan's giant cells - Giant cells may contain 2 inclusion: • Schaumann bodies: round calcified laminated • Asteroidbodies: star- shaped	Rounded or oval aggregation of: • Many epitheloid cells • Langhan's giant cells • Surrounded by: Mantle of lymphocytes & fibroblasts • Cental caseous necrosis: structureless, finely granular, eosinophilic, contain nuclear depris

11) Thrombophlebitis and thrombophlebosis

SEPT 2013

	Phlebothrombosis	Thrombophlebitis
Def.	Thrombus without inflammation.	Thrombus with inflammation
Cause	Virchow triad: - slow circulation - ↑PLT - damage of intima	- Septic: OM, p. sepsis, appendicitis, piles - Non septic: radiation, chemical
Comp.	Detach → Massive Pulmonary Embolism **"If occlude large br."**→ acute Rt HF" → pulm. Infarction **"if small br."** →red infarction	- Septic emboli → Pyaemic abscess - Non septic→ infarction

12) Empyema and pyaemia

	Empyema	Pyaemia
Def	Accumulation of pus in hollow organs	multiple small abscesses in organs dt fragmentation of thrombus into septic emboli
Sites	e.g. pleura , gall bladder , appendix	e.g. lung in puerperal sepsis , liver in infected piles

13) Compare between acute and chronic graft rejection

	Acute	Chronic
Time	Within days-weeks of transplantation or in months or even years later	Within months – years
Mechanism	-Caused by both cellular & humoral response Mechanisms Types -Acute Cellular Rejection : T cells destroy graft parenchyma by cytotoxicity & delayed hypersensitivity reaction. -Acute Vascular Rejection : T cells & antibodies damage graft vasculature.	T cell reaction & secretion of cytokines
Pathologic Features	-Interstitial Mononnuclear cellular infiltrate -Vasculitis → Infarction or Renal cortical atrophy	-Arteriosclerosis -Interstitial Fibrosis -Loss of renal parenchyma -No response to standard immunesuppression regimens.

SECTION 3 | GIVE REASONS FOR EACH OF THE FOLLOWING

1) Occurrence of leukocytosis in acute inflammation.

JUNE 2012

- TNF and IL-1 accelerate the release of cells from the BM reserve pool
- Production of colony stimulating factor (CSF) by activated macrophages leads to increase BM production of leucocytes to compensate for those lost in inflammatory reaction.

2) Infection with staph aureus cause localized suppurative inflammation

SEPT 2012

- *Staph. Produce* coagulase enzyme which convert fibrinogen to fibrin that help in localization of the infection.

3) Infarction of the lung is of hemorrhagic type

SEPT 2012

- Has a dual circulation from bronchial and pulmonary artery
- Has loose tissues

So, lung permits blood flow from the patent vessels into the infracted area.

4) In primary TB, the draining lymph nodes are usually affected.

SEPT 2012

- Because the patient has not developed immunity yet so:
  - The phagocytic activity is minimal
  - Many bacilli are carried to the draining LNs in order to be a sensitized person

5) In Actinomycosis there is no lymphatic spread

MIDYEAR 2015

-Because the causative organisms (Actinomyces Israelii) are Large gram positive filamenous bacteria which can't enter the lymphatic vessels.

6) Bilharziasis cause Ammonia encephalopathy

MIDYEAR 2015

- ↓ Conversion of ammonia to Urea → ammonia reach systemic circulation.
- Escape of ammonia through open porto systemic shunts → systemic circulation of the brain → encephalopathy & Coma.

7) Severe bleeding in Patient with Disseminated Intravascular Coagulopathy (DIC)

MIDYEAR 2015

Because there is sudden onset of widespread of fibrin thrombi in microcirculation → Rapid consumption of platelets & coagulation proteins with activation of fibrinolytic mechanisms → serious bleeding.

8) Hypercalcemia & Hypercalcuria in Sarcoidosis

MIDYEAR 2015

Due to production of active Vitamin D by MCs in Sarcoidosis granulomas → Increased calcium absorption.

SECTION 4 | EXPLAIN EACH OF THE FOLLOWING

1) Role of C3b in acute inflammation (phagocytosis)

- C3b is an important opsonin that coat the micro-organisms and target them for phagocytosis
- Leucocytes bind and ingest the micro-organism via specific cell receptor that recognize the opsonin

2) Formation on inflammatory exudate is an important phenomenon in acute inflammation.

JUNE 2011

- Dilute bacterial toxins
- Brings antibodies to inflammatory area as bacteriolysins , agglutinins , opsonins that destroy , fix and coat the injurious agent
- Contains fibrinogen that changes into insoluble fibrin which forms a network on which :
  - Leucocytes move in the direction of the organism
  - Help in localization of the infection by surrounding the inflamed area
  - Fibroblast move to start healing process
- Contain neutrophils which kill the organism

3) Role of interferon gamma (IFN- γ) in pathogenesis of tuberculosis.

JUNE 2012

- Macrophage activation and differentiation into epithelioid cells that have increased phagocytic and microbicidal activity

4) Pathogenesis of caseation in tuberculosis

JUNE 2013

- CD4+TH1 facilitate the development of CD8+ cytotoxic T cells which can kill tuberculosis-infected macrophages directly causing caseation necrosis

5) Right side heart failure may be a complication of pulmonary bilharzial lesion.

JULY 2011

- Dead worms cause severe allergic inflamm. and vascular necrosis "Verminous- Pneumonia"
- Ova causes vascular lesions which produce necrotizing arteriolitis, fibrous thickening of the intima with narrowing of the lumen and obliterating arteriolitis.
- These vascular lesions lead to pulmonary hypertension and this lead to Right side heart failure

6) Organ specificity in blood spread of malignant tumors (give 4 examples).

JUNE 2012

- Due to organ tropism which is expression of adhesion molecules or chemokines by tumor cells whose receptors is expressed on the endothelium of the target organs.

7) Occurrence of tumor angiogenesis.

SEPT 2012, SEPT 2013

- Essential for growth of 1ry tumor > 1 mm & facilitate metastasis.
- Controlled by balance between:
  - Angiogenic factors as VEGF released by tumor & TNF α by MC.
  - Inhibitor of angiogenesis as angiostatin.

8) Lower limb venous thrombosis following major operation.

MIDYEAR2012

a- Slowing of the cirulation as a result of the lack of muscular activity from prolonged recumbence and shallow breathing, both decreasing the venous return to the heart

b- Damage to the intima due to pressure on calf muscles by recumbency which also impairs blood flow.

c- Increased- number- and- adhesiveness of platelets.

9) Mutation of TP53 may be associated with malignant transformation.

MIDYEAR 2012

- P53- is- the "guardian of genome"
  - ↓DNA damage.
  - ↑DNA repair by G1 arrest.
  - control- expression- of- genes- as- DNA- repair, cellular aging, apoptosis- and- gene- involved- in- cell- cycle- as- RB. .

- So homozygous loss of P53 → unrepaired DNA → Malignancy.

10) Patients with xeroderma pigmentosa are at increased risk of developing sunlight induced squamous cell carcinoma (2m).

SEPT 2012

- Due to- inherited- mutation- of- DNA- repair- genes

11) Some virus infections may be associated with carcinogenesis.

JUNE 2012

- HPV →Sq. cell papilloma, cervical carc. By certain types
- EBV → nasopharyneal carc. & Burkitt's lymphoma
- HBV & HCV→ HCC "hepatocellular carc."
- HTLV→Lymphoma &Leukemia

12) The prognosis of basal cell carcinoma is better than that of squamous cell carcinoma.

- Basal- cell carcinoma- is- locally malignant tumor – with- no distant metastasis "no lymphatic or bl. spread".
- But squamous cell carcinoma- can- se- can- send- distant metastasis- by- blood- or lymphatic- so- has- a- worse- prognosis.

SECTION 5 | COMPLETE THE FOLLOWING

1) Granulation tissue is composed of ..... , ... & .....
JUNE2013

- Newly formed capillaries
-Macrophages
-Fibroblastic reaction

2) Oxygen free radicals lead to cell injury by causing .....&.....
JUNE 2014

-Lipid peroxidation
-Oxidation of proteins
-DNA damage
-Cytoskeletal damage

3) The extra cellular matrix is composed of ..... , ... & .....
JUNE 2014

-Fibrous structural protein e.g. collage & elastin
-Water hydrated gel e.g. proteoglycan & hyaluronan
-Adhesive glycoprotein e.g. fibronectin

4) In acute inflammation, the chemical mediators involved in causing pain are .....&.....
JUNE 2014

-Prostaglandins
-Bradykinin

5) In cases of regeneration, the role of transforming growth factor β is ...& ..
JUNE 2014

-Chemotactic for PMNs,macrophages,lymphocytes,fibroblasts and smooth muscle cells
-Stimulate tissue inhibitor of matrix metalloproteinase (TIMP) synthesis
-Regulate integrin expression and other cytokines
-Inhibit production of matrix metalloproteinase & keratinocytes proliferation

6) Acquired immunodeficiency syndrome is characterized by depletion of ...
JUNE2014

-T4 helper cells

7) - Rupture of hydatid cyst cause .....
MIDYEAR 2015

Anaphylactic Shock.

8) - CVC of lung characterized microscopically by ..... , while Gamma-Gandi nodules are characteristic feature of .....
MIDYEAR 2015

Heart failure cells, CVC spleen.

9) - Blastoma is ...
MIDYEAR 2015

Group of malignant tumors which arise from embryonal or partially differentiated cells that has not yet fully developed and occurs more in infants and children.

10) Hamartoma is .....
MIDYEAR 2015

Tumor like malformation present as a mass of disorganized but mature tissues normally present in affected part but often with one element predominating.

11) From the characteristics of malignant cells ..... , ... , ... , ...
MIDYEAR 2015

Loss of polarity, pleomorphism, Hyprchromatism, Anisonucleosis.

SECTION 6 | ENUMERATE

12) Histological types of carcinoma. Give an account on squamous cell carcinoma (Sites, gross and microscopic picture).

MAY 2008

➢ **Histologic types:**

a- Surface epith.
- Sq.Cell.Carcinoma = epidermoid carc.
- Basal Cell.Carcinoma= rodent ulcer
- Transitional- cell- carcinoma

b- Glandular epith.
- Adenocarcinoma
- mucinous = mucoid
- Signet ring carc.

➢ **Squamous cell carcinoma:**

	SCC = epidermoid carc.
Site	- Any st. sq. epith as skin - Sq. metaplasia as before
Gross	• Papule→fungating + ulcer • Malignant Ulcer "everted edge, necrotic floor & hard base dt infiltration"
MP	- Irregular **masses of** sq. epith. **e criteria** of malignancy... - **Cell nests or pearls**: The center of the masses show esinophilic keratin. sq. cells Accord. to **Border's classification** if 50-75% of masses show nest, the tumor is well diff., but if <25% of masses show nest, it is poorly diff.
Spread	**- Direct:** "infiltration", **- Lymphatic, blood:** "late"

End of The Book ☺